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Dear Joshua:

I have got the job to answer your letter of Dec.9, since I have been the last to read Boyd's paper.

It is hard to explain lysis inhibition on the basis of a phage heterogeneity because of this: if one infects with very low multiplicity using T2r+, without diluting before the lysis, one does get lysis inhibition, presumably due to the readsorption of the first phage to be liberated onto the cells which have not lysed yet. If phage were heterogeneous, the first phage to appear should be of the rapidly lysing type and therefore could not lysis-inhibit.- Also, if a no matter how large multiplicity of phage is adsorbed in a very short period, no lysis inhibition occurs.

According to old Doermann's paper a first single infection with r followed by r^+ does not lysis inhibit. Still these data should be checked with better techniques than those which Doermann could use at that early time. On the plates, one would say that a r^+ plaque inhibits adjacent r plaques, but it is hard to interpret what is going on on the plates.

As to Boyd's paper, I have been puzzled a lot, in trying to find an interpretation able to explain all his data. If it is only question of heterogeneity of the phage (and "predominance" of the lysogenic one), one should expect to find within a certain range of multiplicity a linear relationship between induced lysogenic and phage input. His data are too scarce to show such relationship.-If it is only a question of multiplicity, one should not find this relationship.- From table III, assuming this last hypothesis to be the good one, one can calculate that approximately 2.5-3 particles are necessary to make a cell lysogenic. Then, in the experiments of table I, (for instance in columns 10^7 and 10^8 phages/cc) one does not understand why there is such a big loss of cells. Since they get infected presumably after they have gone thru a few divisions, one should expect under these conditions that a multiple infection of 2 or 3 phage particle on a cell adjacent to one which met one phage particle and was lysed, is a very common occurrence, following in most cases a lytic infection.

It is quite possible that there is some relation between induced lysogenesis and lysis inhibition. Still, from what we know on lysis inhibition, I have the impression that the delaying block occurs at a later stage than one would expect for a block supposed to make the multiplication of the prophage indefinite (see for instance the data of Cohen on DNA in multiply infected cells).

I had news of you from Seymour. I might be able sometime

to win my natural laziness and come up to visit you.

Best regards,
sincerely

Joe